

CASE REPORT

Listeriosis in an immunocompetent patient after diagnostic colonoscopy

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Abstract

We present an 80-year-old female with type II diabetes (well controlled) who presented to the emergency department with a hemoglobin of 6.5 mg/d consistent with iron deficiency anemia (IDA). As part of the workup for IDA, she had an esophagogastroduodenoscopy (EGD) and colonoscopy. EGD was unremarkable. Colonoscopy revealed a mass occupying about 50% of the circumference of her descending colon suspicious for malignancy, which was biopsied. Thirty-six hours later, she developed fevers; blood cultures grew *Listeria monocytogenes*. Workup to identify the source of bacteremia was negative for other sources of infection. Due to the temporal relationship, the development of bacteremia was attributed to the disturbance of the gastrointestinal tract possibly from recent biopsy of the colonic mass. She was treated with penicillin for a total of about 4 weeks with complete resolution of symptoms and clearance of bacteremia. She had a transverse colectomy 6 weeks later with surgical pathology of the lesion showing intramucosal adenocarcinoma. This case represents a rare complication of colonoscopy and is novel because our patient was not immunocompromised as previously reported in other cases.

INTRODUCTION

Colonoscopy is an extremely common medical procedure, which represents the cornerstone of the detection and prevention of colorectal cancer. While colonoscopy is considered to be very safe, there are inherent risks associated with this procedure, including the uncommon incidence of infection [1]. Although exceedingly rare, there are documented cases of patients with systemic infections caused by *Listeria monocytogenes* infections following mechanical disturbance of the GI tract [1–6]. These systemic infections with listeria are referred to as listeriosis. Previous cases describe patients with underlying risks for systemic infection, often patients with inflammatory bowel disease on chronic immunosuppressive medication. In this report, we

examine the case of an immunocompetent patient with listeriosis following colonoscopy with biopsy.

CASE REPORT

The patient is an 80-year-old female with a medical history of hypertension, diabetes mellitus type II, embolic stroke, atrial fibrillation, percutaneous pacemaker, enterococcus endocarditis secondary to an epidural abscess (resolved), ischemic colitis diagnosed with colonoscopy 7 years prior, and left hemiarthroplasty. Her diabetes was well controlled with metformin, and her hemoglobin (Hgb) A1c was 5.9% 10 months prior and 6.3% on the day of admission. She was referred to the emergency department

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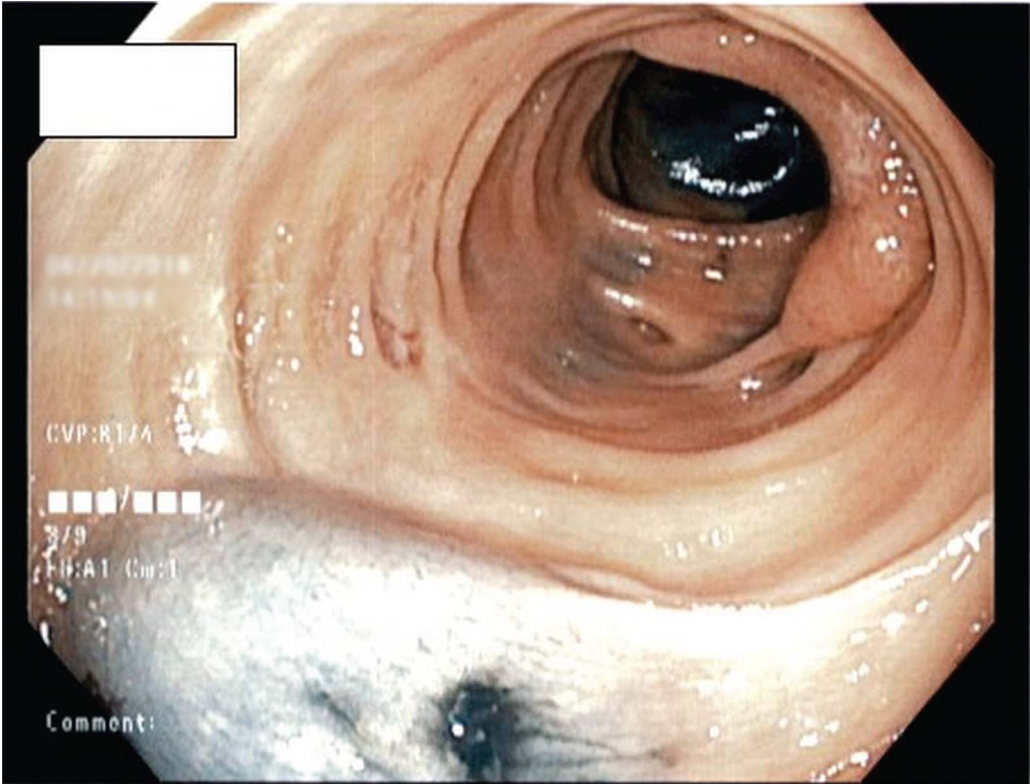


Figure 1: Colonoscopy showing a suspicious lesion occupying about 50% of the circumference of the descending colon.

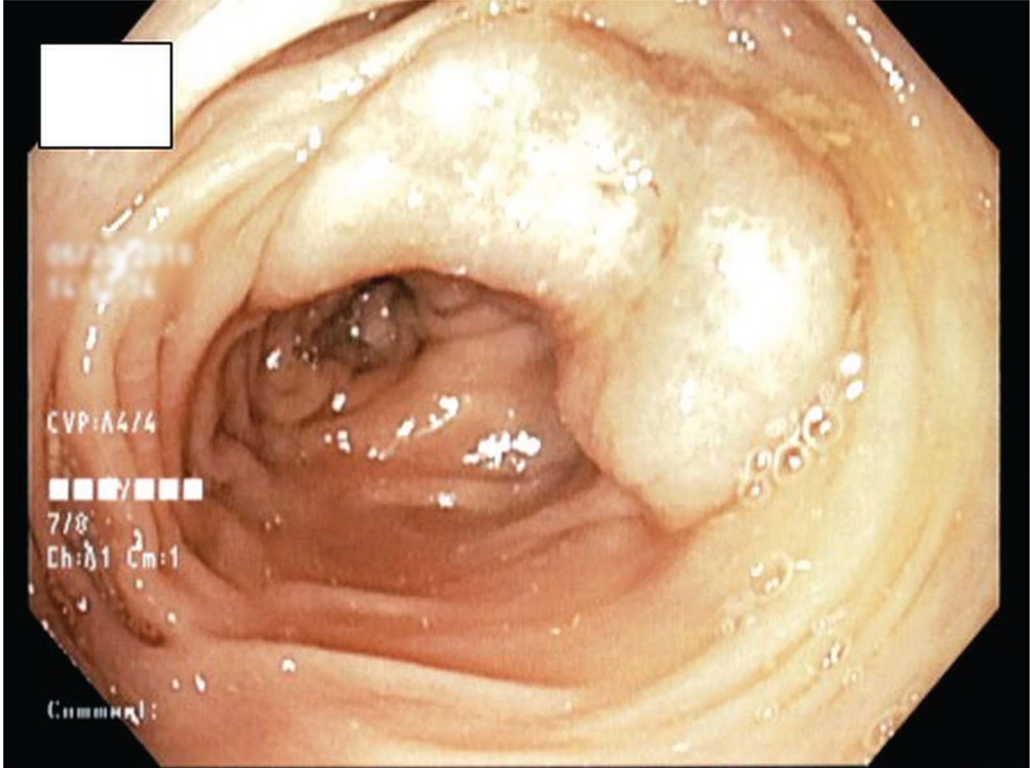


Figure 2: Malignant circumferential lesion seen in the descending colon on colonoscopy.

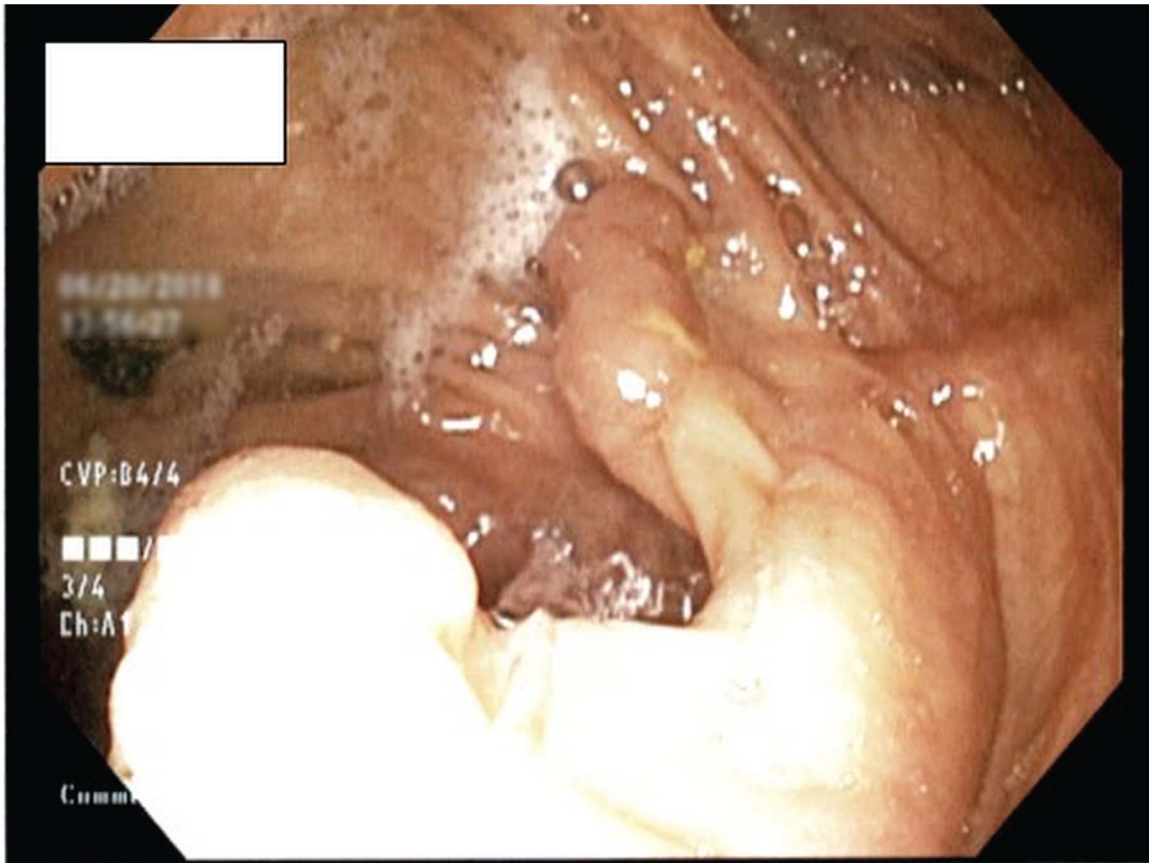


Figure 3: Colonoscopy showing a close-up view of the suspicious-looking mass seen in the descending colon.

(ED) by her primary care physician for Hgb of 6.5 mg/dl on routine screening lab work. In the ED, her vitals were stable and the physical examination was unremarkable. Investigations revealed iron deficiency anemia. No gross source of bleeding was identified. She denied melena, hematochezia, weight loss, or non-steroidal anti-inflammatory drug use. She received 1 unit of packed red blood cells (PRBCs) upon arrival. Following administration of PRBCs, her Hgb improved to 8.0 mg/dl and remained relatively stable.

The gastroenterology service was consulted and recommended that she undergo esophagogastroduodenoscopy (EGD) and colonoscopy to diagnose the cause of her iron deficiency anemia. She initially refused colonoscopy, so EGD was performed. EGD showed no evidence of neoplasia or mucosal abnormality. Following the results of the EGD, she consented to have a colonoscopy. Colonoscopy was performed 2 days following EGD and revealed the following: two 15-mm sessile polyps in the cecum, moderate diverticulosis of the ascending and transverse colon, one 6-mm polyp of the descending colon, and a 35-mm suspicious-looking mass occupying 50% of the circumference of the descending colon (Figs 1–3). Snare cautery was performed to remove the three smaller polyps. Biopsies were taken from the mass (Fig. 4), and spot ink tattoo was performed proximal and distal. All polyp biopsies demonstrated tubular adenoma, with biopsies from the mass representing high-grade dysplasia.

Approximately 36 hours after colonoscopy, she had a single episode of fever. Her temperature was 38.3°C (101°F) with no other signs of sepsis. Initially, the decision was made to obtain infective workup, which included blood and urine cultures, and

observe for reoccurrence of fever or additional signs of infection. She had several episodes of fever the following day with a maximum oral temperature of 39.5°C (103.1°F) recorded. Gram stain from the blood culture revealed gram-positive bacteria. She was empirically started on intravenous vancomycin for coverage of gram-positive bacteria, as well as metronidazole for anaerobic coverage given recent endoscopy. Blood cultures subsequently grew *L. monocytogenes*. Infectious disease (ID) service was consulted. Repeat blood cultures remained positive for *L. monocytogenes* for a total of three consecutive days. Computed tomography of the thorax, abdomen, and pelvis was negative for metastatic lesions or another source of infection. She did not exhibit any signs of central nervous system (CNS) infection. She was not on immunosuppressive medications and had no previous history of being immunosuppressed. Ultrasound of the pacemaker was negative for loculated collections. Transthoracic echocardiogram did not show any vegetation, but a mitral valve posterior leaflet calcification was seen. Subsequent transesophageal echocardiogram ruled out endocarditis. Antibiotics were deescalated to i.v. ampicillin upon identification of the organism and return of sensitivities.

At time of discharge, due to the history of previous enterococcus infective endocarditis, percutaneous pacemaker *in situ*, left hemiarthroplasty and presumed immunocompromised state from malignancy given the presence of colonic mass, a decision was made by the ID physician to continue i.v. penicillin for 3 weeks following the last positive blood culture. Six weeks following discharge, she underwent transverse colectomy with surgical pathology of the lesion showing intramucosal adenocarcinoma.

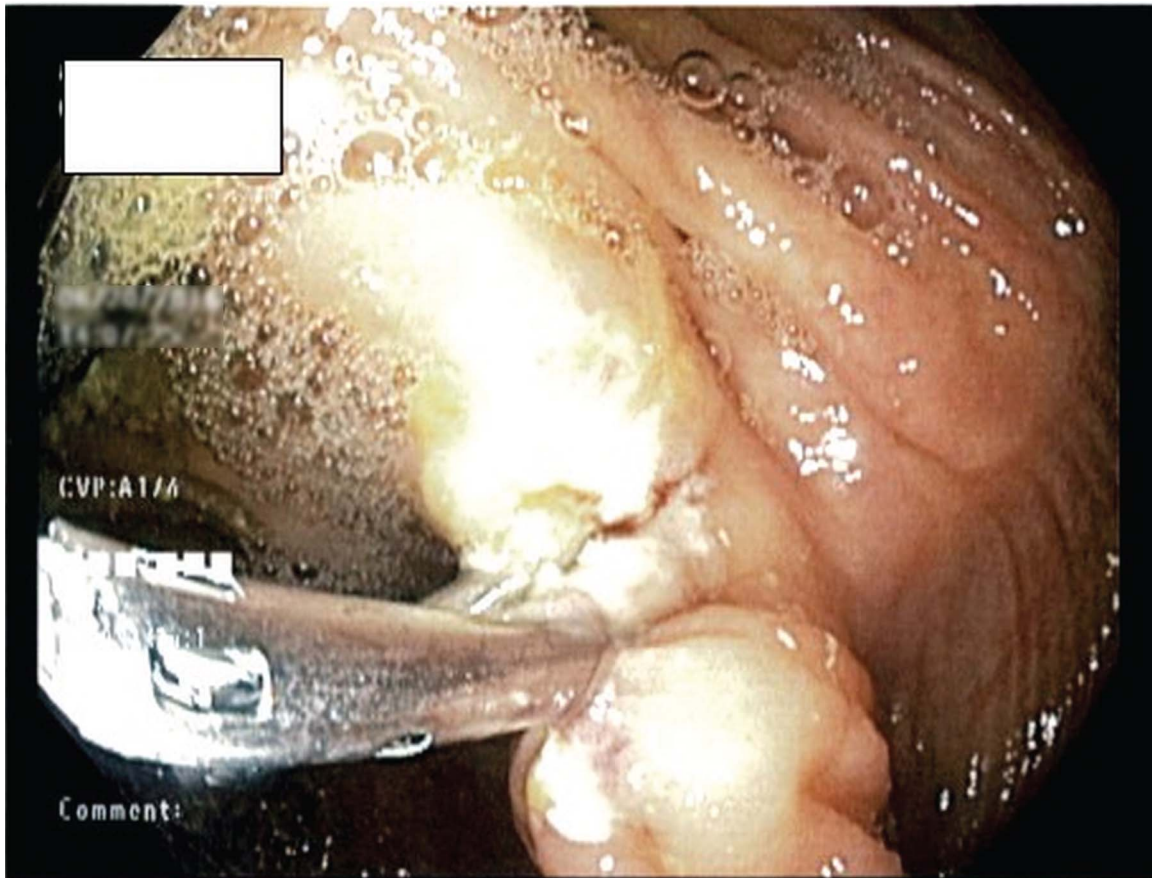


Figure 4: Colonoscopy showing endoscopic biopsy of suspicious-looking lesion in the descending colon.

DISCUSSION

Post-operative infections following endoscopy are divided into two categories: endogenous and exogenous. Endogenous infections are the more common of the two and occur as a result of the patient's normal gastrointestinal flora leading to infection. Exogenous infections are spread from patient to patient with contaminated endoscopes. Listeriosis is considered to be an endogenous infection given that it is a colonizer of the gastrointestinal tract [1, 3]. There is a wide range of reported post-operative bacteremia following colonoscopy, ranging from 0% to 25%, with a mean of 4.4% in small sample studies [1, 3]. While this may seem like a frighteningly common occurrence, it is important to remember that the majority of these studies focused on transient bacteremia, as opposed to symptomatic infections [1]. Symptomatic infection following colonoscopy remains a rare occurrence and is most commonly found in immunocompromised patients [1].

Colonization of the alimentary tract with *Listeria* has been seen in as many as 0.8–3.6% of immunocompetent individuals [4, 7]. These individuals, however, rarely present with symptoms related to this colonization [1]. The immunocompetent who do experience symptoms due to *Listeria* generally present with febrile, non-bloody gastroenteritis. Systemic infection can present multiple different ways, including bacteremia, endocarditis, osteomyelitis, septic arthritis, and CNS infection, with meningitis being the most common presentation [5, 6, 8]. The choice and duration of treatment for listeria infections are largely dependent on clinician interpretations of the data and

preferences as there are no controlled clinical trials to establish a preferred drug or duration of therapy [9].

Prior reported cases of listeriosis following colonoscopy had been predisposed with underlying risks, which lent to the susceptibility of widespread disease. Some of the described predisposing conditions for listeriosis following colonoscopy include cirrhosis, systemic malignancy, HIV, and previous organ transplant or inflammatory bowel disease on chronic immunosuppression [1, 2]. The proposed pathogenesis for listeriosis following colonoscopy is that the procedure creates breaks in the mucosa, allowing *Listeria* to cross this barrier. This, coupled with impaired cellular immunity, leads to infection. Due to the temporary relationship in the case reported, the development of bacteremia was attributed to the disturbance of the gastrointestinal tract from recent colonoscopy with biopsy of the colonic mass. No workup regarding endoscope contamination was performed, as it was unlikely to be from endoscopic contaminant and more likely endogenous as previously reported.

The mass found on colonoscopy was demonstrated to be intramucosal adenocarcinoma on surgical pathology and did not represent invasive malignancy, thus, this should not cause systemic immunosuppression. Considering this patient's risk factors, this case represents a unique presentation of a patient with listeriosis following colonoscopy in an immunocompetent patient and emphasizes the need to maintain a broad differential, which should include listeriosis, even in immunocompetent patients presenting with signs of infections following a colonoscopy.

CONFLICT OF INTEREST STATEMENT

None declared.

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ETHICAL APPROVAL

No institutional review board (IRB) approval was needed. Exempt from IRB approval.

CONSENT

Patient has given written consent for writing and publication for this case report.

GUARANTOR

J.B., S.K. and I.E. are the guarantors of the article.

CONTRIBUTION

J.B., S.K. and I.E. were involved in writing, reviewing, and approving the final manuscript for publication. The authors thank Christopher Cooper, MD, Infectious Disease Physician, Department of Internal Medicine, Michigan State University, for his contribution to this manuscript.

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